British Heart Journal, 1970, **32**, 477–482.

Correlation between radiological chest findings and systemic haemodynamics in human arterial hypertension

Rune Sannerstedt, Sven Paulin, and Ed Varnauskas
From the Department of Medicine I and the Roentgen Diagnostic Department I, Sahlgrenska Hospital, University of Göteborg, Göteborg, Sweden

Radiographic chest findings from 57 men, aged 17 to 64, with untreated systemic arterial hypertension of varying severity were related to systemic haemodynamics, both at rest and during standardized exercise in the sitting position. The haemodynamic findings were compared with corresponding data from 59 normotensive male volunteers. In classifying the radiographic findings into four different groups, not only the heart size but also the appearance of the aorta and left ventricle were taken into account.

While there was only a poor correlation between calculated heart volume and resting mean brachial artery pressure or systemic vascular resistance, the classification system used resulted in a good separation for haemodynamic functions of patients with normal chest films, those with elongated and widened aorta, and those with signs of left ventricular hypertrophy. On the other hand, there was no definite difference in the functions studied for hypertensive patients with left ventricular hypertrophy and those with, in addition, signs of left ventricular dilatation.

Hypertensive men with normal chest radiographs stood out as characterized by increased heart rate and cardiac output at rest, while the systemic vascular resistance was not different from that of the controls. Already the presence of an elongated and widened aorta changed this haemodynamic pattern to that characteristic of established arterial hypertension with normal resting heart rate and cardiac output, and increased systemic vascular resistance.

Considerably better information on the haemodynamic events in systemic arterial hypertension is possible to achieve with a thorough evaluation of the radiological appearance of the aorta and left ventricle, rather than with estimation of the heart size only.

A direct correlation between the radiologically measured heart volume and the height of the blood pressure has been reported (Humerfelt, 1963). Similarly, a successively impaired prognosis with increasing heart size has been described in patients with arterial hypertension (Sokolow and Perloff, 1961; Tominaga and Yoshida, 1967), as also a positive correlation between heart volume and pulmonary artery pressure, and a negative one between heart volume and left ventricular work or stroke work indices (Varnauskas, 1955). On the other hand, many authors have found radiography of the heart to be a poor guideline in the evaluation of hypertensive patients and in severity of the disease (Rasmussen and Thingstad, 1939; Ehrström, 1948; Kleinfeld and Redish, 1952; Josephsen, 1956; Braun and Hornung, 1965; Ramirez and Garcia Pont, 1965; Sokolow *et al.*, 1966; Zweifler and Thomas, 1968). These statements have, however, usually been based only on analysis of the transverse or postero-anterior diameter of the cardiac silhouette or on the calculated heart volume, without taking into consideration the appearance of the aorta and/or the left ventricle.

Received 29 December 1969.

In judging the effect of arterial hypertension on the appearance of the chest radiograph, the configuration of the left ventricle and the aorta must, however, also be considered. Thus, prominence of the aortic knob in patients with hypertensive cardiovascular disease has been found to be the single most significant finding in various diseases leading to increased transverse cardiac diameter (Gustafson and Friedenberg, 1965), and an elongation of the left ventricle has been stated to be the first abnormal finding in arterial hypertension (Holzmann, 1952).

Studies relating radiological signs of left ventricular and aortic enlargement to the haemodynamic events in arterial hypertension have hitherto been lacking. Therefore, an attempt has been made to classify chest *x*-rays from 57 men with untreated arterial hypertension and to relate the radiological findings to systemic haemodynamics obtained both at rest and during exercise, taking into account not only the heart size but also the appearance of the left ventricle and the aorta. The haemodynamic data are compared with those from 59 normotensive men studied under identical conditions.

Subjects and methods

The group of 57 hypertensive men studied has been presented in detail elsewhere (Sannerstedt, 1966). Their ages ranged from 17 to 64 years (Table). All of them had auscultatory blood pressure recordings repeatedly above 150/90 mm.Hg. Based on an extensive clinical work-up, 48 patients were classified as essential hypertension in various stages of the disease and 3 as possible renovascular hypertension. The remaining 6 patients had parenchymatous renal disease. None of the patients was taking antihypertensive drugs at the time they were studied.

The control group consisted of 59 men aged 16 to 59 with casual blood pressures below 150/90 mm.Hg and no signs of cardiovascular or respiratory disease (Table). The majority, 41 altogether, were healthy volunteers. The other 18 were patients at the hospital with mild signs and symptoms, if any, of disease.

Standardized teleradiographs in the frontal, sagittal, and left oblique standing positions were obtained in all hypertensive patients. The films were analysed by one of us (S.P.) without having access to any information other than the clinical diagnosis of arterial hypertension. The heart volume was calculated according to Jonsell (1939), and the upper normal limit was set at 450 ml./m.^{2} body surface area without taking into account the variation with age shown by *inter alia* Humerfelt (1963). An aortic width above 35 mm. was considered abnormal in subjects below 50 years old, provided the body surface area was less than 1·90 m.^{2} Otherwise no attempt was made to correct for the influence of age and body size, described by several authors (Ungerleider and Gubner, 1942; Strehler, 1965). The control group was not studied radiologically.

Four subgroups of hypertensive men were formed, based on the appearance of the left ventricle and the aorta, and the heart size, as follows:

X-ray group 1: normal radiography of the chest.
X-ray group 2: moderate elongation of the aorta, and/or aortic width greater than 35 mm. provided body surface area less than 1·90 m.^{2} and age below 50 years.
X-ray group 3: considerable elongation of the aorta, and/or signs of left ventricular hypertrophy.
X-ray group 4: *x*-ray group 3 + signs of left ventricular dilatation, and heart volume greater than 450 ml./m.^{2} body surface area.

TABLE *Age and body surface area (mean ± SE, and mean difference from the controls)*

Group	*No.*	*Age (yr.)*		*Body surface area (m.^{2})*	
		Mean ± SE	*Mean diff.*	*Mean ± SE*	*Mean diff.*
Controls	59	33·9 ± 1·5		1·935 ± 0·021	
Hypertensive men					
X-ray group 1	10	32·4 ± 4·3	−1·5	1·848 ± 0·042	−0·087
X-ray group 2	12	48·3 ± 2·6	+14·4*	1·875 ± 0·027	−0·060
X-ray group 3	29	46·9 ± 1·6	+13·0*	1·966 ± 0·031	+0·031
X-ray group 4	6	49·5 ± 2·1	+15·6*	2·030 ± 0·063	+0·095

* = p value for the difference from the controls < 0·001.

Hypertensive patients showing radiological signs of cardiac failure with left atrial enlargement and pulmonary congestion, or substantial radiological changes not referable to hypertensive cardiovascular disease, have not been included in the present analysis.

The examination procedure and techniques used for analysis of haemodynamic data have been described in detail elsewhere (Sannerstedt, 1966). The functions studied in the morning in the fasting state were: heart rate, intra-arterial blood pressure, cardiac output using a dye-dilution technique with bromsulphalein as the indicator and with intermittent sampling of arterial blood, and oxygen consumption with sampling of expired air in a Douglas bag for subsequent gas analyses according to the method of Scholander. From the data obtained, systemic vascular resistance in arbitrary units was calculated.

The first measurements were made after one half-hour of rest, with the subjects sitting comfortably in an arm-chair. Then, one or two periods of exercise were performed sitting on an electrically braked, variable-load bicycle ergometer. Each period lasted for 10 to 12 minutes, with a resting period of 15 to 20 minutes in between, and the level of exercise was adopted to the subject's tolerance. The cardiac output was measured during the tenth minute of exercise.

The data from both the hypertensive and control subjects were fed into a computer (Facit EDB3) for statistical analysis of the results. The response to exercise was related to increase in oxygen consumption, and the differences between the values at rest and during exercise for the various functions were converted to express the difference per rise of one litre in oxygen consumption. In addition, the data from the control series both at rest and during exercise were subjected to multiple regression analyses with respect to influence of age, weight, height, and body surface area. On comparing data for the hypertensive groups and the control series, the haemodynamic variables for the hypertensive groups were corrected for age and physical characteristics, whenever significant regressions ($p < 0.05$) between these data were found. The Student t test was used for testing the significance of differences between the hypertensive groups and the controls.

Results

In Fig. 1 the total heart volume measured radiologically has been plotted against mean brachial artery pressure and systemic vascular resistance at rest. The poor correlation between these two functions and the heart volume is readily seen. A slight positive correlation seems to be present, when the heart volume is expressed as an index instead of the total volume, but there is still a considerable scattering of the values for mean pressure and vascular resistance.

Using the present classification system, a progressive increase in resting brachial artery pressure with increased *x*-ray grouping was obtained, as seen in Fig. 2. The separation between the patient groups is most obvious for *x*-ray groups 1 and 2, and between groups 2 and 3, while the average figures for groups 3 and 4 are almost identical. On exercise, *x*-ray group 1, in comparison with the other hypertensive patient groups, stands out as showing less increases in the mean and diastolic brachial artery pressures than the controls, the difference in response of the diastolic pressure being significant.

The patients in *x*-ray group 1 were also characterized by a significantly higher heart rate at rest than the control subjects (Fig. 3). All three other patient groups had resting heart rates not different from that of the controls. The responses of heart rate to exercise tended to be excessive in *x*-ray groups 1–3, the differences from the controls being significant for groups 2 and 3.

The resting cardiac output was significantly higher in *x*-ray group 1 than in the controls, and from the increased resting level this patient group raised the cardiac output significantly less during the following exercise (Fig. 3). The other patient groups did not differ from the controls as regards the cardiac output, either in resting values or in responses to exercise, but a tendency to a pattern similar to that for group 1 may be seen to be present also in hypertensive patients classified as *x*-ray group 2.

Hypertensive men with normal radiography of the chest (*x*-ray group 1) and the control subjects had on an average identical systemic

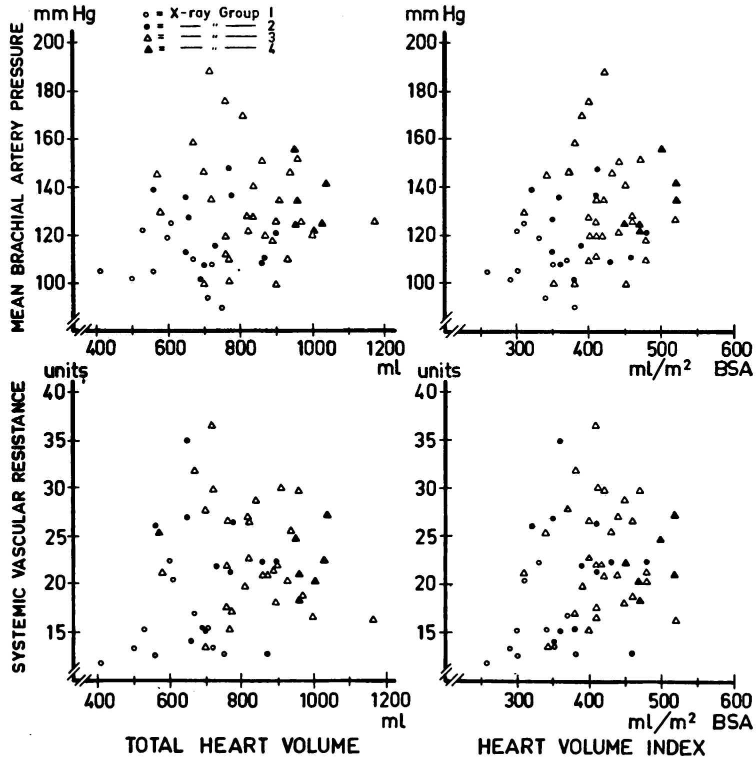


FIG. 1 *Relation between mean brachial artery pressure and systemic vascular resistance, and total heart volume and heart volume index.*

vascular resistances at rest, and the decreases in resistance on exercise were not different (Fig. 3). Patients in *x*-ray groups 2–4, on the contrary, had significant increases of resting vascular resistance, and the falls during exercise were more conspicuous than in the controls, the differences being significant for groups 3 and 4.

Discussion

While the various patient groups did not differ significantly from controls in body surface area, the mean ages of groups 2–4 were significantly higher than those of the controls. To minimize such bias, the data from the controls were subjected to multiple regression analysis and, before comparing patients and controls, the haemodynamic findings for the hypertensive groups were corrected for the influence of age and physical characteristics. We therefore feel that the differences found between the patients and the controls can be considered to be due to factors other than any differences in age and body characteristics.

The poor correlation between heart volume

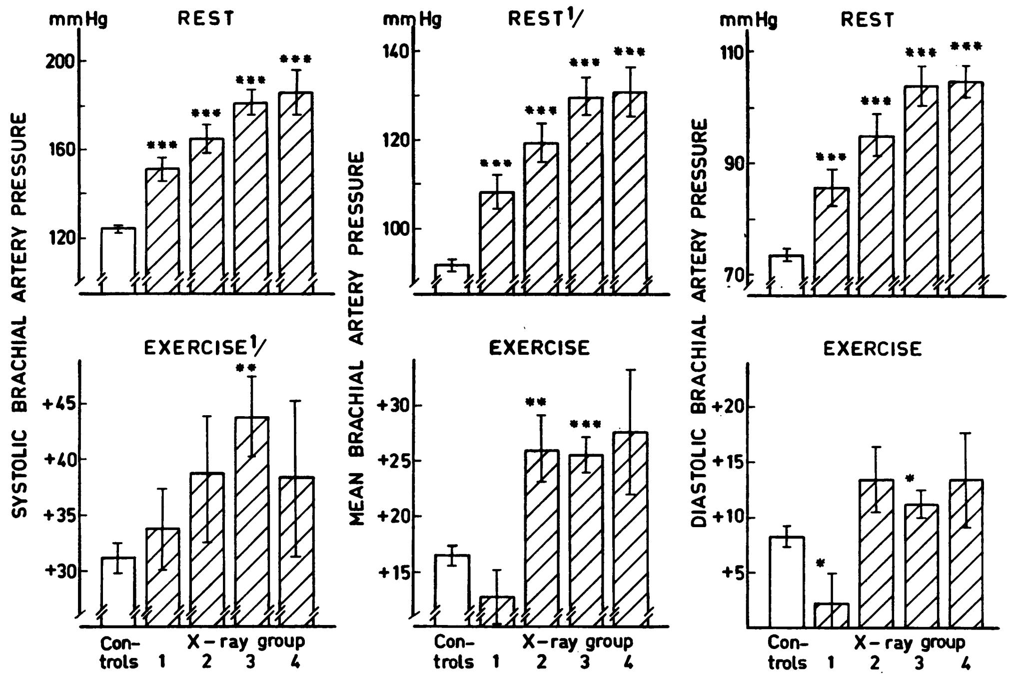
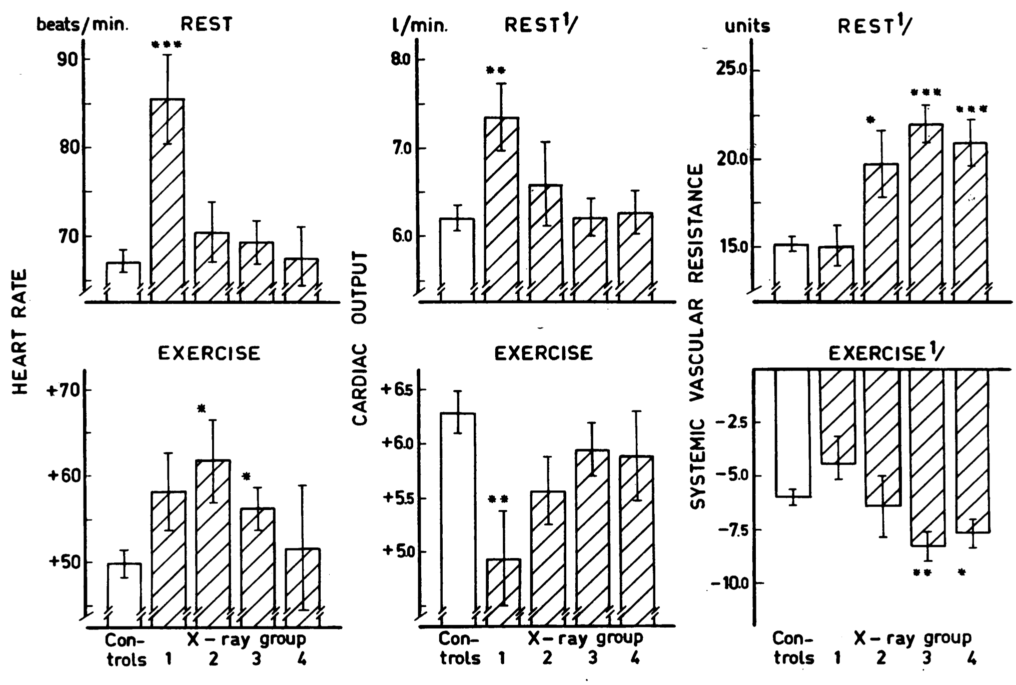


FIG. 2 *Brachial artery pressures at rest and the response to exercise (mean ± SE). The latter is presented as the average difference in pressure per one litre increase of oxygen consumption.* *, **, *and* *** *indicate p values of* < 0·05, < 0·01, *and* < 0·001, *respectively, for differences between the patient and control groups.* ¹/ *Mean values in the patient groups adjusted for the influence of age.*

FIG. 3 *Heart rate, cardiac output, and systemic vascular resistance at rest and the response to exercise (mean ± SE). The latter is presented as the average difference per one litre increase of oxygen consumption. For symbols see Fig. 1.* ¹/ *Mean values in the patient groups adjusted for the combined influence of age and body surface area.*



on the one hand, and mean brachial artery pressure or systemic vascular resistance on the other, shown in the present study, indicates that estimating the heart size only does not reflect to an accurate degree the strain laid on the left ventricle in patients with arterial hypertension. This agrees with the findings of other authors (Rasmussen and Thingstad, 1939; Ehrström, 1948; Josephsen, 1956; Ramirez and Garcia Pont, 1965), who found the transverse diameter of the cardiac silhouette or the calculated heart volume in hypertensive patients to correlate poorly with the height of the blood pressure.

Taking not only the heart volume, but also the radiological appearance of the left ventricle and the aorta into account, as done in the present classification, gave a stepwise increase of the average resting intra-arterial blood pressure with increased grouping of radiographic chest changes. The difference between the average values for *x*-ray groups 3 and 4 was, however, small.

This trend to a good separation between *x*-ray groups 1–3, but not between groups 3 and 4, became more obvious for the resting cardiac output and systemic vascular resistance. Thus, the average resistance was even somewhat less among patients with signs of left ventricular dilatation – *x*-ray group 4, as compared with patients with left ventricular hypertrophy but no dilatation – *x*-ray group 3. This was due to a slightly higher resting cardiac output in group 4, and not to a lower mean arterial pressure. As the response of the cardiac output to exercise was similar in *x*-ray groups 3 and 4, and not significantly different from that of the controls, it might be postulated that the radiographic signs of left ventricular dilatation seen in patients classified as group 4 do not reflect an additional strain on the left ventricle or impending heart failure.

The classification system picked up hypertensive men with normal radiographic findings – *x*-ray group 1 – as differing from the other patient groups, characterized at rest by increased heart rate and cardiac output with a systemic vascular resistance not different from that of the controls, and on exercise a similar or even lower rise in blood pressure than the controls together with a less increase in cardiac output. This haemodynamic pattern is equivalent to that previously described by several authors as occurring in patients with mild, uncomplicated arterial hypertension (Eich *et al.*, 1962; Bello, Sevy, and Harakal, 1965; Finkielman, Worcel, and Agrest, 1965; Sannerstedt, 1966; Julius and Conway, 1967; Lund-Johansen, 1967).

The changes in the haemodynamic pattern from *x*-ray group 1 to group 2 are of special importance, the heart rate and cardiac output at rest in the latter group being the same as those of the controls. It seems therefore as if the presence of an elongated and widened aorta in an otherwise normally sized and configurated heart would make it possible to distinguish patients with established, high resistance-normal output arterial hypertension from those in the early, uncomplicated stage of the disease. However, even if the differences between various groups are statistically significant, there is a considerable overlap between patients and controls for the functions studied. Therefore, the predictive value for the individual patient may be limited.

Nevertheless, the present results indicate that a thorough evaluation of the appearance of the aorta and left ventricle will yield considerably better information on the haemodynamic events in systemic arterial hypertension than estimation of the heart size only.

References

Bello, C. T., Sevy, R. W., and Harakal, C. (1965). Varying hemodynamic patterns in essential hypertension. *American Journal of the Medical Sciences*, **250**, 24.

Braun, H., and Hornung, G. (1965). Zur Grösse des Herzvolumens bei kardiovaskulär kompensierten Hypertonikern. *Archiv für Kreislaufforschung*, **46**, 139.

Ehrström, M. C. (1948). Enlargement of the heart in hypertension. *Acta Medica Scandinavica*, **130**, Suppl. 206, p. 86.

Eich, R. H., Peters, R. J., Cuddy, R. P., Smulyan, H., and Lyons, R. H. (1962). The hemodynamics in labile hypertension. *American Heart Journal*, **63**, 188.

Finkielman, S., Worcel, M., and Agrest, A. (1965). Hemodynamic patterns in essential hypertension. *Circulation*, **31**, 356.

Gustafson, J. E., and Friedenberg, M. J. (1965). Evaluation of left heart disease by statistical analysis of aortic parameters and transverse cardiac diameter. *American Heart Journal*, **69**, 479.

Holzmann, M. (1952). Erkrankungen des Herzens und der Gefässe. In *Lehrbuch der Röntgendiagnostik*, 5th ed., p. 2679. Ed. by H. R. Schinz, W. E. Baensch, E. Friedl, and E. Uehlinger. Georg Thieme Verlag, Stuttgart.

Humerfelt, S. Bj. (1963). An epidemiological study of high blood pressure. *Acta Medica Scandinavica*, **175**, Suppl. 407.

Jonsell, S. (1939). A method for the determination of the heart size by teleroentgenography (a heart volume index). *Acta Radiologica*, **20**, 325.

Josephsen, J. O. (1956). Hypertension and the size of the heart. (In Norwegian.) *Nordisk Medicin*, **56**, 1335.

Julius, S., and Conway, J. (1967). Hemodynamics in patients with transient blood pressure elevation. *Journal of Laboratory and Clinical Medicine*, **70**, 1026.

Kleinfeld, M., and Redish, J. (1952). The size of the heart during the course of essential hypertension. *Circulation*, **5**, 74.

Lund-Johansen, P. (1967). Hemodynamics in early essential hypertension. *Acta Medica Scandinavica*, **183**, Suppl. 482.

Ramirez, E. A., and Garcia Pont, P. H. (1965). Relation of arterial blood pressure to the transverse diameter of the heart in compensated hypertensive heart disease. *Circulation*, **31**, 542.

Rasmussen, H., and Thingstad, R. (1939). Cardiovascular changes in essential hypertension with special reference to the electrocardiogram in hypertension. *Acta Medica Scandinavica*, **101**, 237.

Sannerstedt, R. (1966). Hemodynamic response to exercise in patients with arterial hypertension. *Acta Medica Scandinavica*, **180**, Suppl. 458.

Sokolow, M., and Perloff, D. (1961). The prognosis of essential hypertension treated conservatively. *Circulation*, **23**, 697.

——, Werdegar, D., Kain, H. K., and Hinman, A. T. (1966). Relationship between level of blood pressure measured casually and by portable recorders and severity of complications in essential hypertension. *Circulation*, **34**, 279.

Strehler, E. (1965). Individuelle Norm der Aortenweite. *Zeitschrift für Kreislaufforschung*, **54**, 571.

Tominaga, T., and Yoshida, M. (1967). Prognosis of essential hypertension. *Japanese Circulation Journal*, **31**, 55.

Ungerleider, H. E., and Gubner, R. (1942). Evaluation of heart size measurements. *American Heart Journal*, **24**, 494.

Varnauskas, E. (1955). Studies in hypertensive cardiovascular disease with special reference to cardiac function. *Scandinavian Journal of Clinical and Laboratory Investigation*, **7**, Suppl. 17.

Zweifler, A. J., and Thomas, J. (1968). Cardiac enlargement in essential hypertension. *Circulation*, **38**, Suppl. 6, p. 26.